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A Case Report of a child with hallucination and aggressive behavior due to AntiNMDAR encephalitis

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Abstract:

Background: Anti-NMDAR encephalitis is an immune-mediated neuroinflammatory disease characterized by autoantibodies against the GluN1 subunit 2B (NR2B)/NMDA subunit 2A (NR2A) subunits of the NDMA receptor in the hippocampus, causing the symptoms.

Case Report: We report a case a13-year-old girl, previously healthy, presented with a history of auditory and visual hallucinations; she saw some of her family members in the room who were not present, and she fought with them; she said they wanted to fight me, attack me, please stop them. She is pitting her tongue, moving it constantly inside and outside her mouth (orofacial dyskinesia), and making abnormal movements with her lips for one day. On the second day of admission, the patient developed aggressive behavior, such as pitting her hands, pulling her hair, throwing objects, seeing people not around, screaming, repeating words, and falling asleep.

Result: The patient initially received ceftriaxone and acyclovir for ten days, olanzapine, and benzodiazepine regularly. The trial of the Prednisolone dose was followed by methylprednisolone, thiamine, biotin, and pyridoxine. EEG showed many Delta brush waves that suggested anti-NMDAR autoimmune encephalitis and NMDA Receptor Abs CSF was 1:16; Glutamate receptor (Type NMDA) IgG abs are detectable in cases of limbic encephalitis. The plan was to start a plasma pheresis with plasma protein for five days, followed by IVIG and Rituximab infusion after virology and quantifiers, which came negative.

Conclusion: Anti-NMDAR encephalitis can be easily diagnosed using serum or CSF sample testing. An index of suspicion should be raised in children presenting with personality changes, abnormal movements or postures, seizures, autonomic instability, or hypoventilation. Management may also be clinically challenging as it involves treating both the cause and the symptoms. A continuous follow-up by the rehabilitation and neurology team is essential. Interdisciplinary communication and collaboration are quintessential to good patient outcomes with this condition.

Keywords: Anti-NMDAR encephalitis (anti-NMDAR), behavioral change, EEG, psychosis.

Background:

Following the first report of pediatric anti-NMDAR encephalitis in China in 2010, anti-N-methyl-d-aspartate receptor (anti-NMDAR) encephalitis is now recognized more frequently and commonly in the pediatric population. It is the second most common etiology for acute demyelinating encephalitis after mixed disturbance encephalitis, surpassing all viral etiologies for encephalitis(1,2).

Anti-NMDAR encephalitis is an immune-mediated neuroinflammatory disease characterized by autoantibodies against the GluN1 subunit 2B (NR2B)/NMDA subunit 2A (NR2A) subunits of the NDMA receptor in the hippocampus, causing the symptoms (3).

Despite being recognized only 13 years ago and having an incidence of only 1.5 per million population per year, more than 1000 cases have been reported until now. It affects most age groups and both genders, with more incidences seen in females (75% cases) (4–9).

Anti-NMDAR encephalitis has four clinical stages (10). Prodromal phase (phase 1): this stage is characterized by fever, headache, nausea, vomiting, and upper respiratory tract infection-like symptoms (11–13). The illness phase (phase 2) consists of the psychiatric and neurological phases. During this stage, MRI abnormalities or pleocytosis can be seen, which decreases over several weeks without visible changes in the symptoms (14). Psychiatric phase- This phase lasts for 1 to 2 weeks. Children present with behavioral changes, irritability, tantrums, coma, manic symptoms, behavioral outbursts, sleep dysfunction, and hyperactivity. Neurological phase- This phase lasts for weeks to months.

Children present with seizures (focal, motor, complex partial), dystonia, or status epilepticus(15). Changes in speech and language (mutism and decreased responsiveness) can be seen within weeks. Catatonia, cardiac arrhythmia, autonomic instability, hypoventilation, and uncoordinated respiration occur, which requires ICU admission. Speech dysfunction is much more commonly seen in children than autonomic dysfunction and hyperventilation (12) (16). Motor dysfunction, in addition to the typical seizures, can develop as dyskinetic movements such as orofacial dyskinesia. Young children can have ataxia or difficulty walking and even lose the ability to walk. Recovery phase (phase 3): Recovery has been described in the reverse order of presenting symptoms (14). The slowest to improve are the cognitive and psychiatric functions (10). The patients can enter the recovery phase after a few months of treatment with appropriate immunotherapy and multidisciplinary care. The presence of inflammation on MRI and CSF is
minimal. Antibodies can persist even after complete recovery (17). Late phase (phase 4): Most patients fully recover cognition and behavioral abnormality at hospital discharge (3).
In the previous literature, many cases of Anti-NMDAR were reported worldwide. In
Saudi Arabia, one case involving a child with anti-N-methyl-D-aspartate autoimmune encephalitis presenting with frank psychosis was reported (18). We report a rare case of Anti-NMDAR, presented with auditory & visual hallucinations and aggressive behavior.

Case report:

A 13-year-old girl, previously healthy, presented with a history of auditory and visual hallucinations; she saw some of her family members in the room who were not present, and she fought with them; she said they wanted to fight me, attack me, please stop them. She is pitting her tongue, moving it constantly inside and outside her mouth (orofacial dyskinesia), and making abnormal movements with her lips for one day. On the second day of admission, the patient developed aggressive behavior, such as pitting her hands, pulling her hair, throwing objects, seeing people not around, screaming, repeating words, and falling asleep. The patient was exposed to stress due to final year exams in the school and lack of sleep before admission. Her symptoms were preceded by viral infection. There is no history of fever, abnormal movement, skin rash, vomiting, or weakness. There is no history history of drug ingestion. Her mother says she is smart and has a good school performance. There is no family history of neurological, psychiatric, or autoimmune disease.
On the investigation: The laboratory showed CBC, ESR, CRP: Normal, Chemistry:
Na 129, liver enzymes slightly elevated, Toxicology screening: negative.
CSF analysis: 10 cells, 100% monocyte, glucoué 3.2 mmol/L, protein: not available. Cultures (blood, urine, CSF, throat swab): negative. Ammonia, lactate, TANDEM: Normal. CT & MRI brain without contrast: Normal. The first EEG on the admission was normal. TFT: pending, dsDNA: pending. Serology (brucella, CMV): negative.
The patient initially received ceftriaxone and acyclovir for ten days, olanzapine PO BID, and benzodiazepine IV regularly. The trial of the Prednisolone dose was followed after methylprednisolone, thiamine, biotin, and pyridoxine. The psychiatrist assessed the patient daily to evaluate and adjust psychiatric medications.
In the third week, the patient still had a slight improvement in her condition. She had aggressive behavior but was not more aggressive than before, and she still had psychosis and auditory and visual hallucinations. EEG and MRI of the brain with contrast were repeated; the EEG showed many Delta brush waves that suggested antiNMDAR autoimmune encephalitis, and the MRI with contrast was normal. LP was done under full aseptic conditions, and samples were sent for CSF cells, chemistry, culture, and samples for CSF anti-NMDA, anti-MOG, and serum samples for antiNMDA; the result showed NMDA Receptor Abs CSF was 1:16 (N= <1:1), Myelin
Oligodendrocyte Glycoprotein ABS (MOG) (BS) was < 1:10 (N=<1:10) and the Comments suggested that: Glutamate receptor (Type NMDA) IgG abs are detectable in cases of limbic encephalitis (often in connection with malignoma/teratoma).
The case was discussed with psychiatrists and neurologists, and both decided on a plan to start a plasma pheresis with plasma protein for five days, followed by IVIG and Rituximab infusion after virology and quantifiers, which came negative. Family counseling was done regarding the benefits and risks of the medications and the expected prognosis, and they accepted it, and consent was signed.
On the first day of plasma Pharesis, the patient was seen as sedated and sleeping. It was challenging to wake her, but she became conscious, looked around, and then went back to sleep. Per her mother, she was always sleeping, awake around midnight, and sitting for around 15 minutes, not talking, only trying to speak but feeling like stuttering speech, then failing to sleep. There were no abnormal movements and no apparent neurological deficit. The patient was on continuous monitoring in the intermediate care room.

On the fifth day of plasma Pharesis, the patient was calm and awake, not agitated, but looked confused. She repeated the wards and was oriented to her name but needed to be oriented to persons or places.

On the patient two weeks later, after plasma Pharesis sessions and IVIG. The patient condition was awake, conscious, oriented to names, times, places, and persons; recognized all her family members; was confused about her condition; still had visual hallucinations, some repetitive talk if she gets excited; obeyed commands but refused to sit or stay in the room. She started physiotherapy sessions. The patient receives one dose of Rituximab according to the protocols.

Patient conditions on discharge: The patient was awake, conscious, and oriented. She returned to her normal mental level and calm and received the psychological assessment and therapy. On neurological examination, the patient had normal power, reflexes, gait, and tone.

On the follow-up, the patient presented to the daycare to receive the second dose of Rituximab two weeks after the first dose. The clinical assessment was done, and she was in normal mental and physical health. A psychiatrist saw the patient, and the plan is to start depakine 250 mg, gradually withdraw benzodiazepine, and decrease the dose of olanzapine to OD with a follow-up next month.

In the future, the patient plans to repeat both EEG and MRI after four months and follow up with psychiatry and neurology.

Discussion:

This case illustrates the successful diagnosis of a 13-year-old girl with Anti-NMDAR Autoimmune Encephalitis. The patient initially received ceftriaxone and acyclovir for ten days, olanzapine, and benzodiazepine regularly. The trial of the Prednisolone dose was followed by methylprednisolone, thiamine, biotin, and pyridoxine. EEG showed many Delta brush waves that suggested anti-NMDAR autoimmune encephalitis and NMDA Receptor Abs CSF was 1:16; Glutamate receptor (Type NMDA) IgG abs are detectable in cases of limbic encephalitis. The plan was to start a plasma pheresis with plasma protein for five days, followed by IVIG and Rituximab infusion after virology and quantifiers, which came negative.

In the previous literature, there is a case reported in Saudi Arabia(18), and we make a comparison between our case and the case of the literature according to the following items (age of the patient, gender, presentation, laboratory finding, radiological finding, management, follow up) in table 1

	Patient 1 (18)	Our Patient
Age and gender	twelve years old, boy	13-year-old girl
Past medical history	developmentally normal, with no family history of psychiatric conditions.	previously healthy, no history of drug ingestion. There is no family history of neurological, psychiatric, or autoimmune disease.
Presentations:	presented 1 week before admission to our hospital with incoherent bizarre speech and delusions where he started to say he is about to die, and express his false beliefs about life, death, heaven and hell and fearing to be left alone in hell. The following day he started having visual hallucinations (pointing and reacting to things that are not there), flight of ideas, and agitation. Then, the boy started to have fever and woke up with poor concentration and disorientation. After a couple of days, other symptoms developed gradually, in the form of Dysphagia to solids then to liquid, Bruxism, abnormal movements in the right hand, and urinary incontinence. One week he developed catatonic and showed a bizarre gait. On examining the head and neck, oro-buccal dyskinesia was observed and dysphagia to solids and liquids, but no cranial nerve affection. His neuromuscular examination showed hypotonia,	presented with a history of auditory and visual hallucinations; she saw some of her family members in the room who were not present, and she fought with them; she said they wanted to fight me, attack me, please stop them. She is pitting her tongue, moving it constantly inside and outside her mouth (orofacial dyskinesia), with making abnormal movements with her lips for one day. On the second day of admission, the patient developed aggressive behavior, such as pitting her hands, pulling her hair, throwing objects, seeing people not around, screaming, repeating words, and falling asleep.

	hyporeflexia and decreased muscle power, in addition to hand automatisms. He then developed attacks of fascio-brachial dystonic seizures.	
Laboratory findings	The cerebrospinal fluid (CSF) antibodies results came positive for anti-NMDA confirming anti-NMDA Autoimmune encephalitis.	and NMDA Receptor Abs CSF was 1:16; Glutamate receptor (Type NMDA) IgG abs are detectable in cases of limbic encephalitis.
Radiological findings:	His EEG showed generalized background slowing which is a picture of encephalopathy and interictal Extreme Delta Brushes, which is a sign seen with anti-NMDA encephalitis.	EEG showed many Delta brush waves that suggested anti-NMDAR autoimmune encephalitis.
Management	we started the empirical therapy for autoimmune encephalitis; intravenous immunoglobulins IVIG 1 g/kg/dose for 2 doses and methylprednisolone 30 mg/kg/day for 5 days. Rituximab (375 mg/m2 weekly for 4 weeks) was added early in the course, as recommended for unresponsive or critically ill patients.	The patient initially received ceftriaxone and acyclovir for ten days, olanzapine PO BID, and benzodiazepine IV regularly. The trial of the Prednisolone dose was followed by methylprednisolone, thiamine, biotin, and pyridoxine. Then the plan was to start a plasma pheresis with plasma protein for five days, followed by IVIG and Rituximab infusion after virology and quantifiers, which came negative.
Follow up	He came for follow-up after 8 months of discharge, walking without assistance, fully verbal, and able to perform all his normal activities with only residual right lower limb weakness.	On the follow-up, the patient presented to the daycare to receive the second dose of Rituximab two weeks after the first dose. The clinical assessment was done, and she was in normal mental and physical health. The patient was seen by a psychiatrist, and the plan is to start depakine 250 mg, gradually withdraw benzodiazepine, and decrease the dose of olanzapine to OD with a follow-up next month.

Table 1: a comparison between our case and the previous cases in the literature

NMDAR is an excitatory glutamate receptor that, when activated, allows the passage of sodium and calcium ions through the channel. Activation occurs by removing the magnesium plug, leading to glutamate and glycine binding to their respective sites (19). Evidence suggests that these antibodies are produced in the CNS by antibodyproducing cells that cross the blood-brain barrier (20). When antibodies bind to NMDAR, it causes internalization of these receptors from the cell surface, leading to receptor hypofunction. The hypoactive receptors failed to cause tonic inhibition on the dopaminergic mesolimbic pathway, resulting in psychosis (19)

First-line treatment involves teratoma resection if present, immunotherapy comprising corticosteroids, intravenous immunoglobulins or plasma exchange, and supportive care (10,21) Second-line treatment using rituximab or cyclophosphamide is most often necessary when the patient does not have an underlying tumor (4,5,11,22) Relapse is more common in patients without a tumor; therefore, continued immunosuppression is recommended for at least one year using drugs such as mycophenolate mofetil or azathioprine (5,6,21) Psychotic and behavioral symptoms are managed using typical or atypical antipsychotics. Severe dopamine blockage can exacerbate dyskinetic movements. The development of neuroleptic malignant syndrome may occur, which can complicate and misdiagnose anti-NMDAR

encephalitis. Quetiapine is the drug of choice for treating psychosis. Valproate works as an excellent mood stabilizer and also offers seizure prophylaxis. Gabapentin and lithium can also be used for mood dysregulation (23–25).

In Conclusion, Anti-NMDAR encephalitis can be easily diagnosed using serum or CSF sample testing. An index of suspicion should be raised in children presenting with personality changes, abnormal movements or postures, seizures, autonomic instability, or hypoventilation. Early diagnosis and treatment are considerable difficulties. Management may also be clinically challenging as it involves treating both the cause and the symptoms. A continuous follow-up by the rehabilitation and neurology team is essential. Interdisciplinary communication and collaboration are quintessential to good patient outcomes with this condition.

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